Armed Forces College of Medicine

AFCM

Neuroscience module

Clinical Integrated Cases
Session 4

Basic Science Principles for Clinical Reasoning

1- **Critical thinking**

2- Introduction to common forms disease

3- Apply basic knowledge in a real clinic scenario.

4- Intern ife long se

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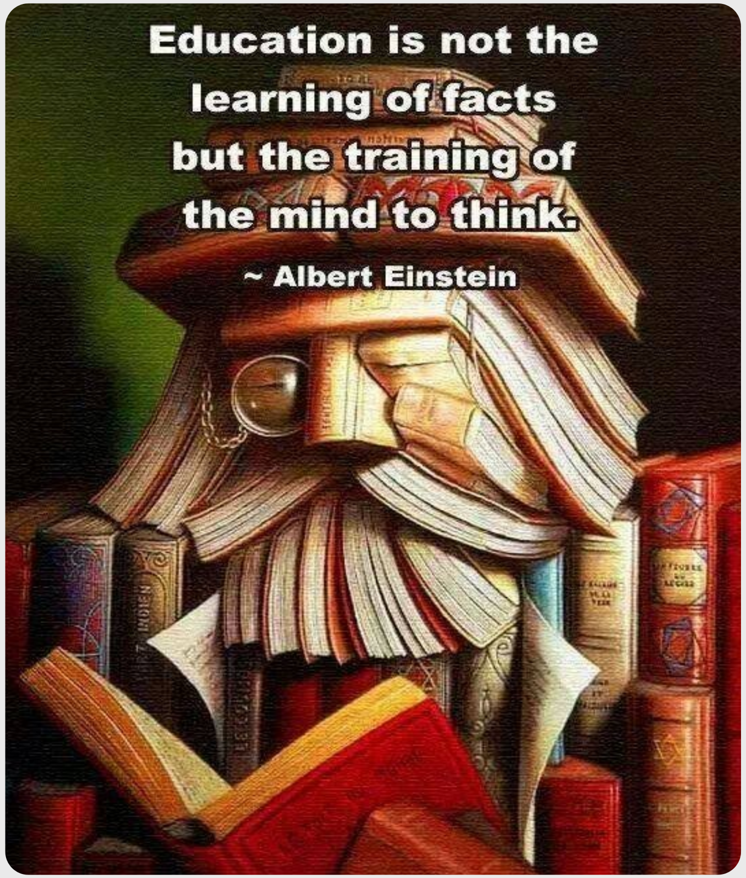
When I hear, I forget
When I see, I remember
When I do, I understand



Education is not the learning of facts but the training of the mind to think.

~ Albert Einstein

Case co-ordinator

Prof. Dr. Manal Hassan Moussa, Lamiaa Foad, Mohamed Edrees

Sharing Departments

- **Anatomy**
- **Neurology Department**
- **Histology Department**
- **Physiology Department**
- **Pharmacology**

I am unable to walk

Monira is a **65** years old **woman**. She lives in Zamalek. Last week, she had **sudden onset of weakness in the left upper and lower limbs**. Her son took her to the hospital emergency room.

She complained of **inability to walk to the bathroom nor even stand.** The condition started **one hour ago.**

The internist knew from the personal history that she **smokes cigarettes with her friends in the club**. He suffers from **chronic hypertension** and **diabetes mellitus**.

I am unable to walk

Vital signs

- Blood pressure was **180/100**,
- Heart rate was **irregular 80** beat/min.

The internist asked for random blood sugar. It was **200 mg/dl**

General examination: No specific abnormalities

I am unable to walk

Neurologic examination

Mental status

Monira is conscious, alert, oriented to time and place and attentive.

Speech: Dysarthria (**Slurred** speech). No dysphagia.

Motor examination

- **No wasting**
- **Weakness** on the left upper movements (inability to do fine distal movements but she can move her shoulders) and lower movements (same weakness degree)
- **decreased tone** on left upper and lower limbs.
- **Diminished deep tendon reflexes** on the left side.
- **Left extensor plantar**.

I am unable to walk

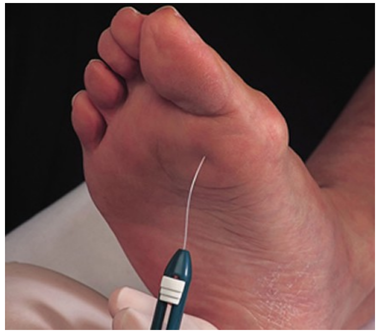
Neurologic examination

Coordination

intact

Sensory examination

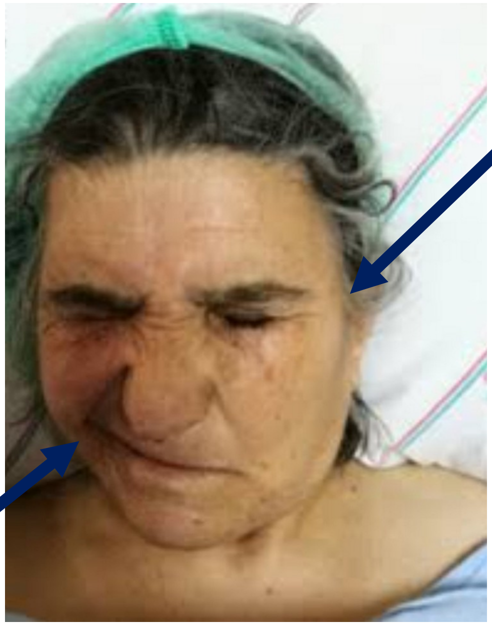
left Hemihypothesia including the face



Cranial nerve examination

Deviation of the angle of mouth to the right side with ability to close her eyes

When she was asked to protrude her tongue, there was **deviation of the tip of the tongue to the left**



Discussion

1- a- List the patient problems

b- What is your provisional diagnosis?

Justify your answer

- **Pyramidal system controlling left side of body**

- **Left facial and hypoglossal nerves (UMN)**

- **Left spino-thalamic tract**

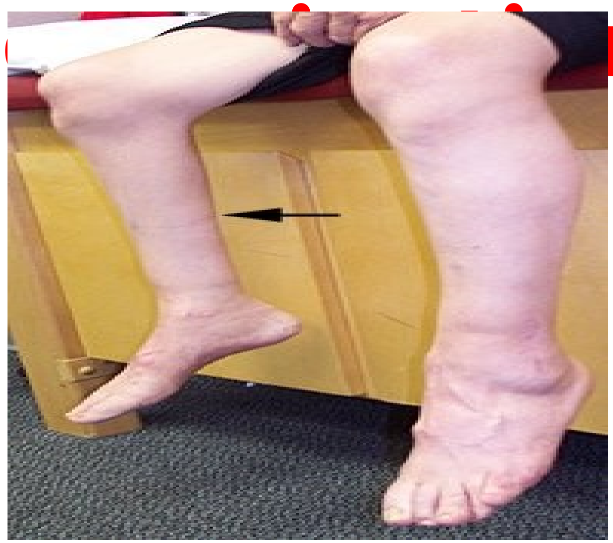
- **Left trigeminal lemniscus**

She complained of **inability to walk to the bathroom nor even stand.**

2- How could you examine the motor system?

1. **Muscle state**
2. **Muscle power**
3. **Muscle tone**
4. **Reflexes**

Motor system

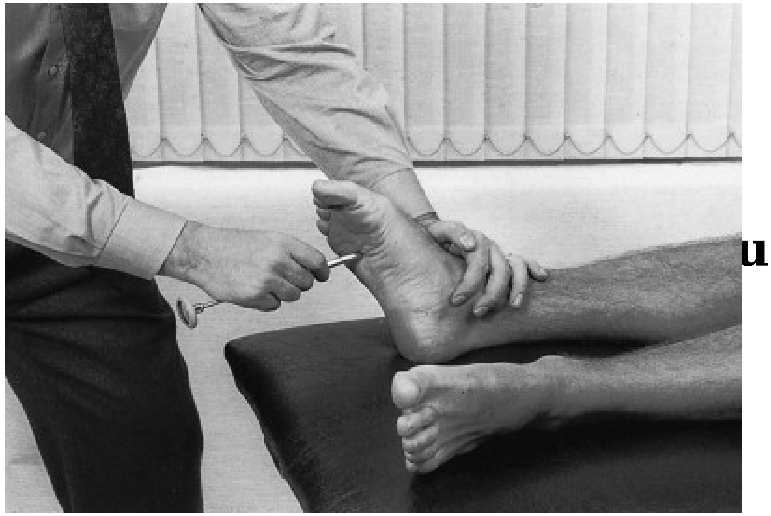


Muscle state

Muscle tone

Muscle power

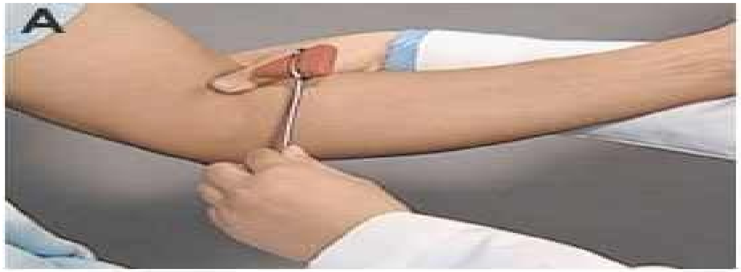
Deep Reflexes

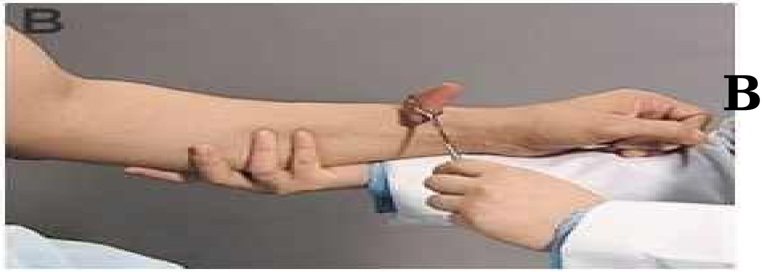


uperficial Reflexes

What are the common reflexes to be examined?

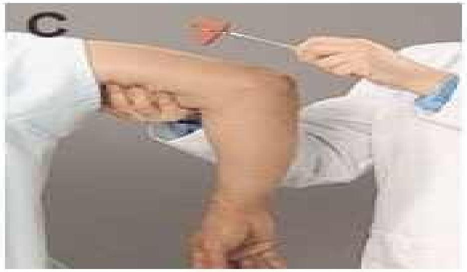
Biceps jerk

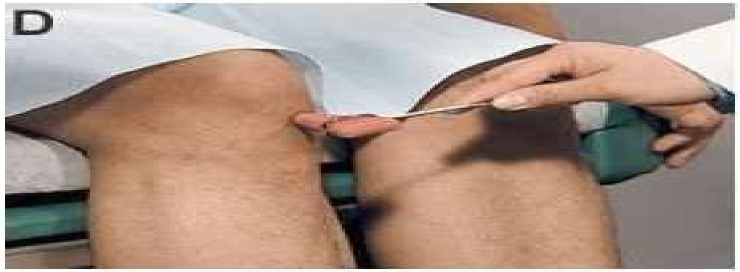




Brachioradialis jerk

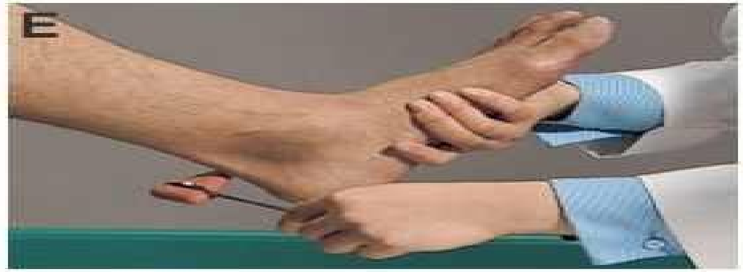
Triceps jerk

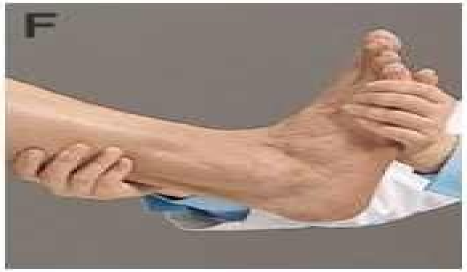




Knee jerk

Ankle jerk





If there is hyperreflexia examine for clonus

Explain the hypotonic weakness

Could it be spastic from the start?

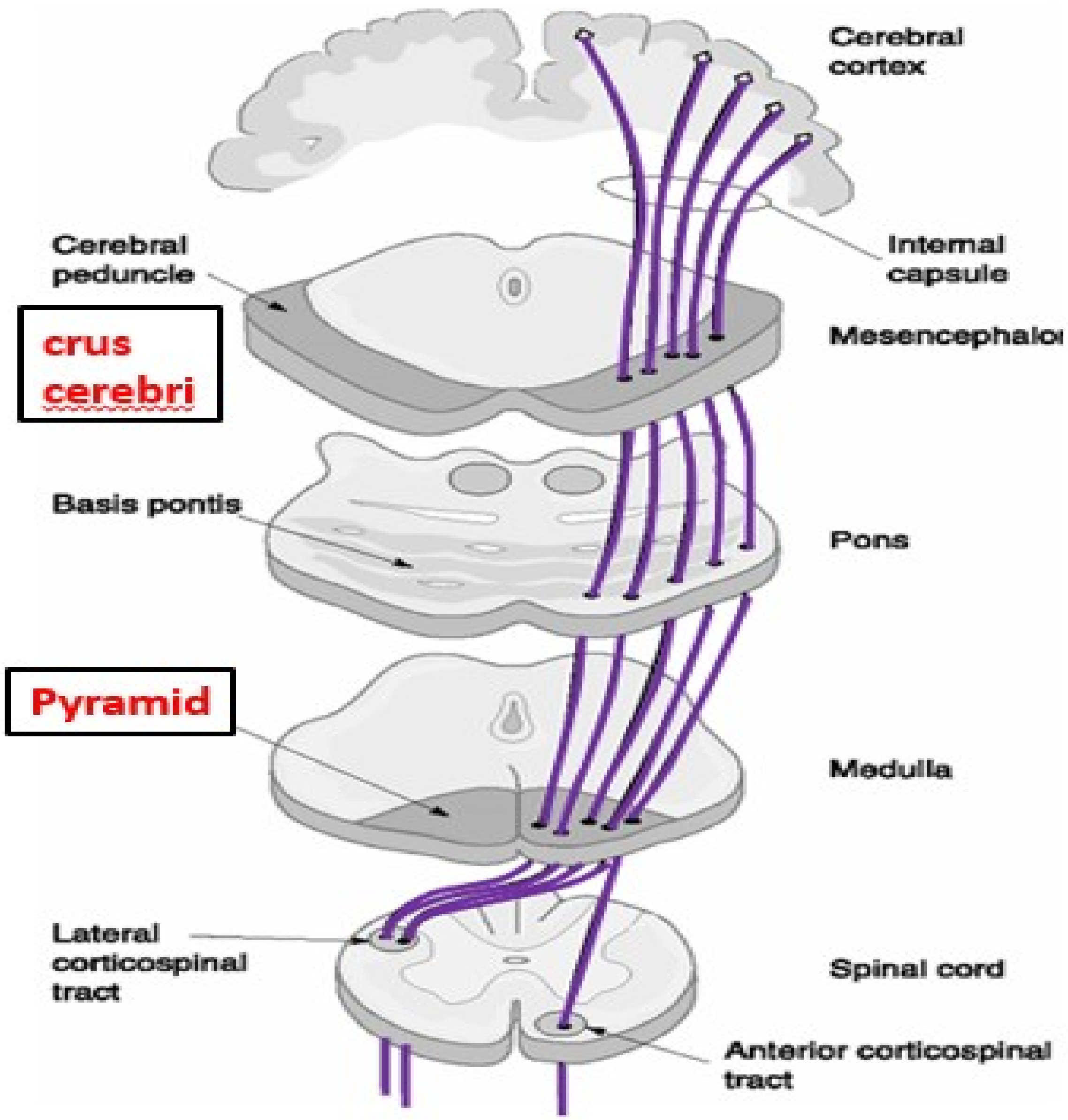
Mostly in basal ganglionic lesions

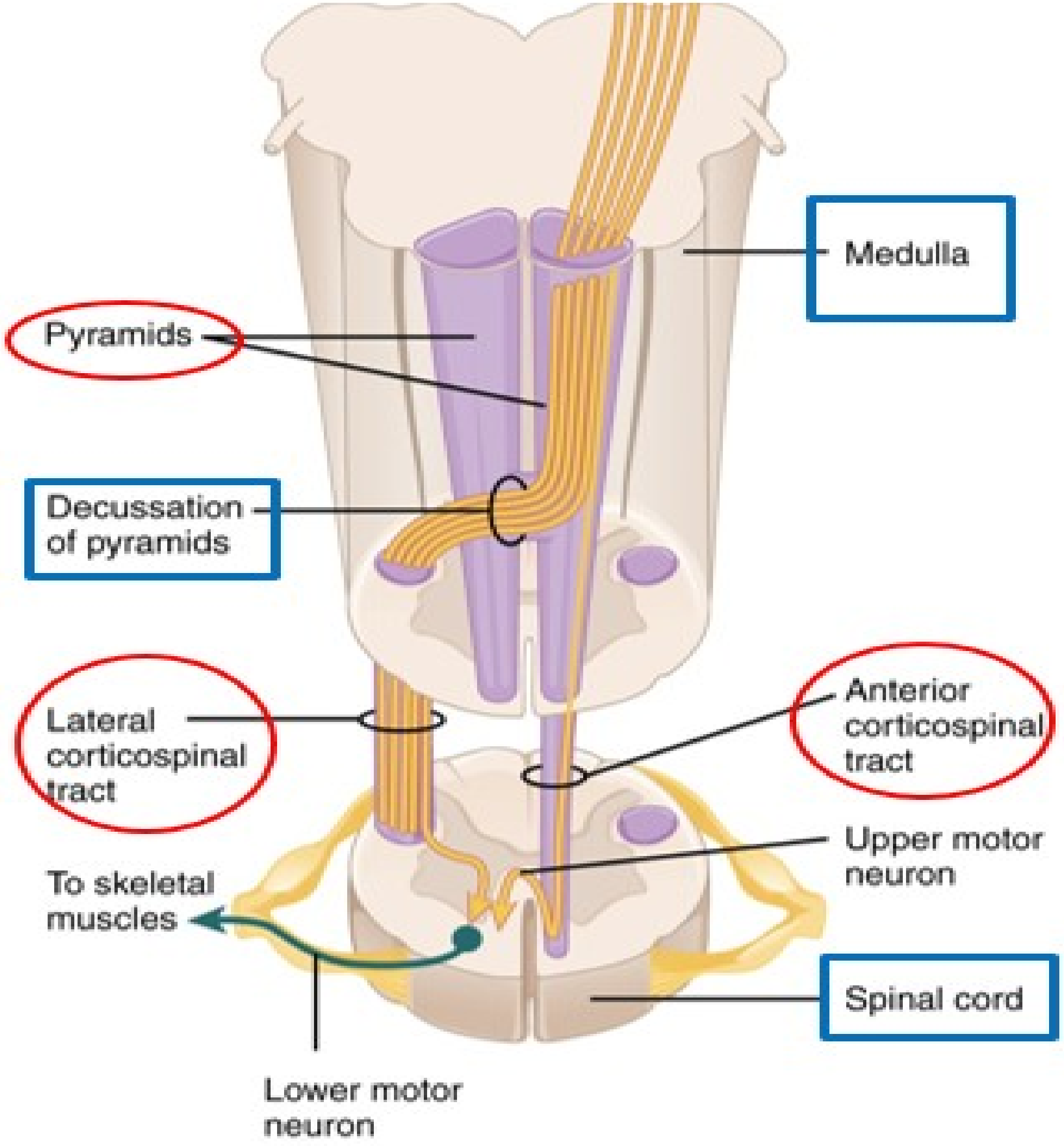
sudden onset of weakness in the left upper and lower limbs

3- What is the tract affected?

Mention its origin, pathway and termination

The affected tract is the

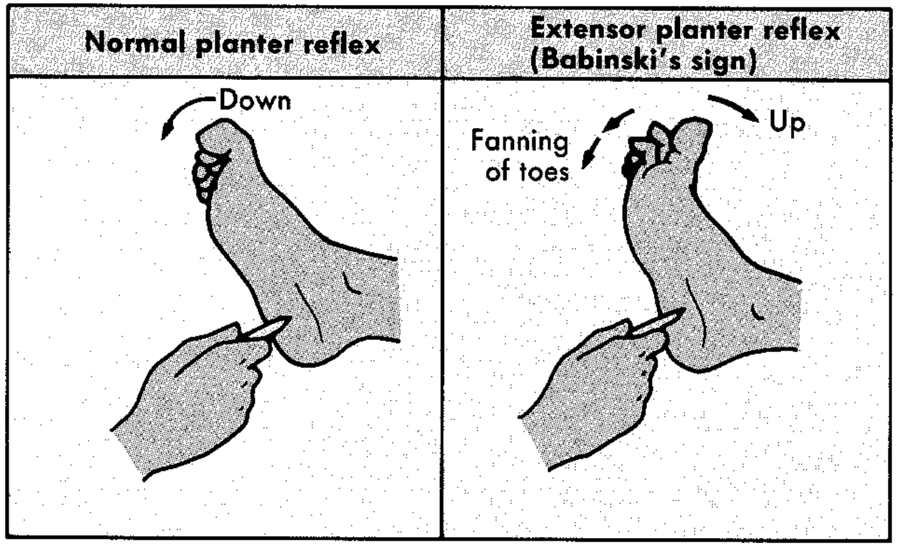


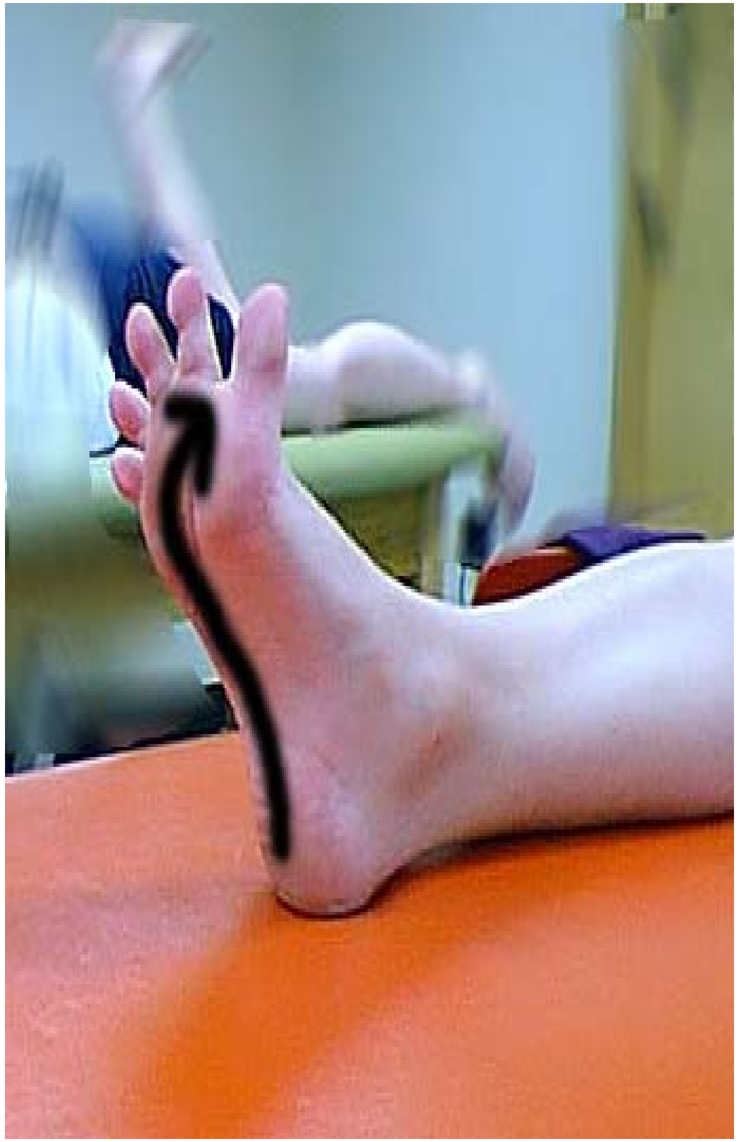


4- What is the test diagnostic of pyramidal le

Planter reflex

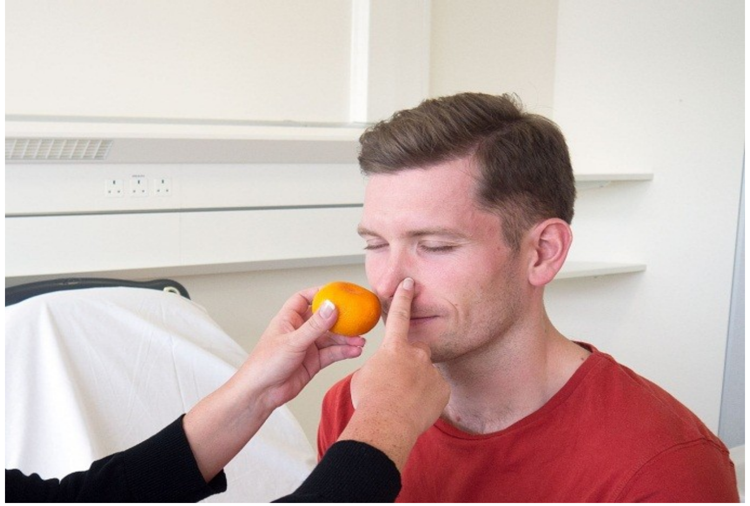
Positive Babinski sign



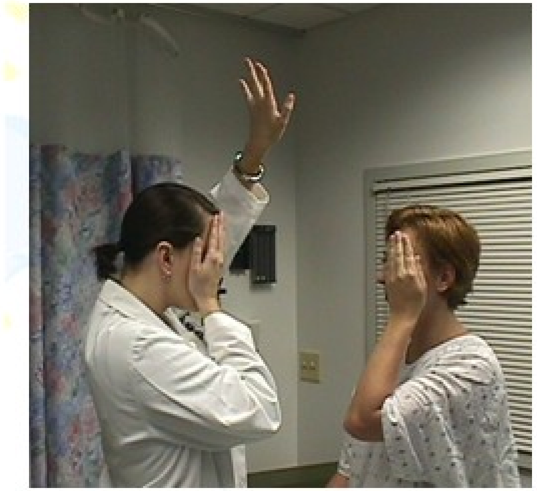


How could you examine the cranial nerves?

Olfactory nerve



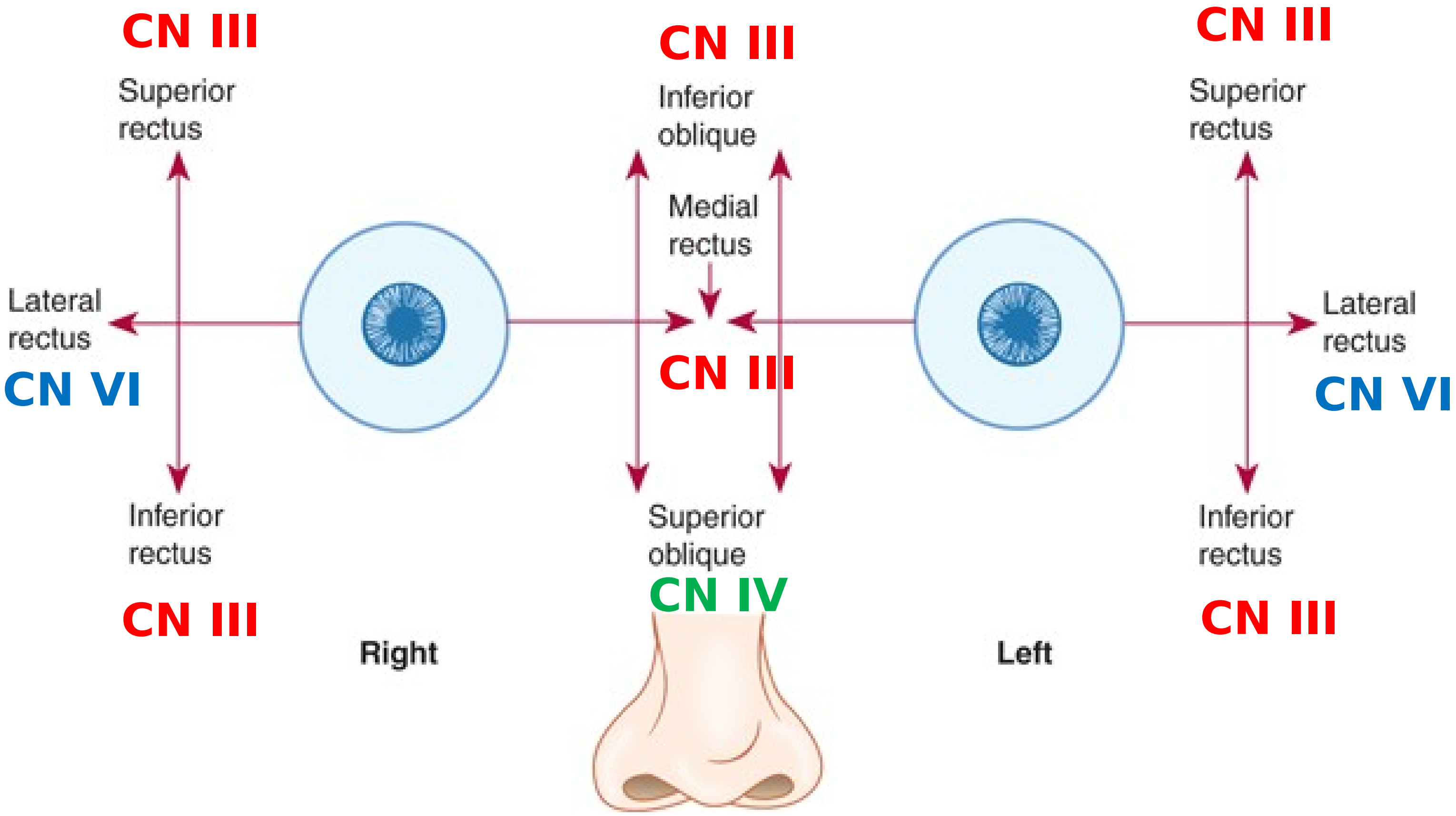
Optic nerve:



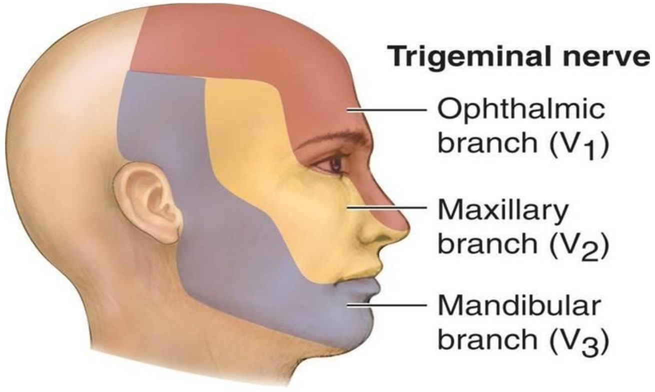
Confrontation visual field exam

Humphrey automated perimetry

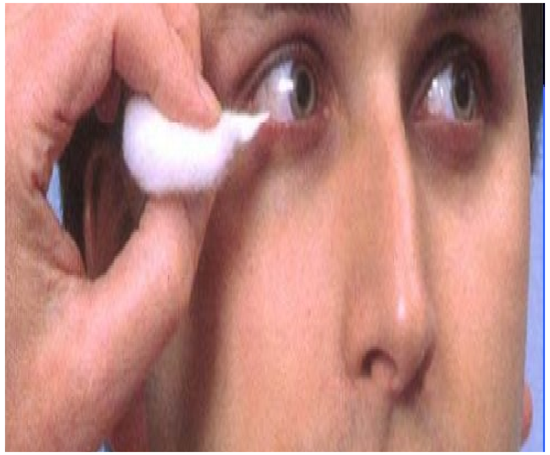
Examine **occulomotor, trochlear, abducent nerves** by examining extra eye movements

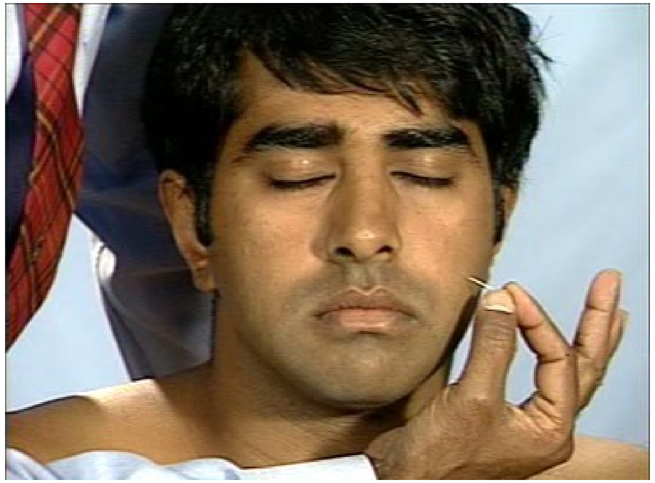


Trigeminal nerve examination

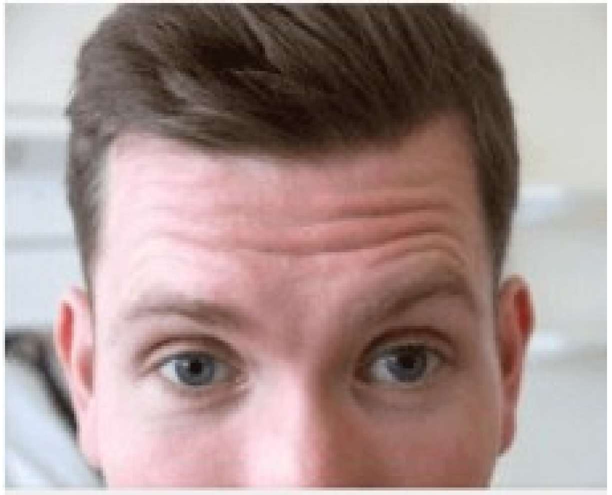


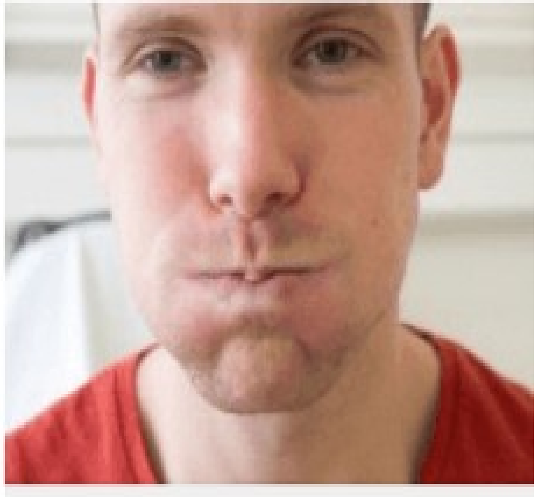
Sensory distribution of trigeminal nerve

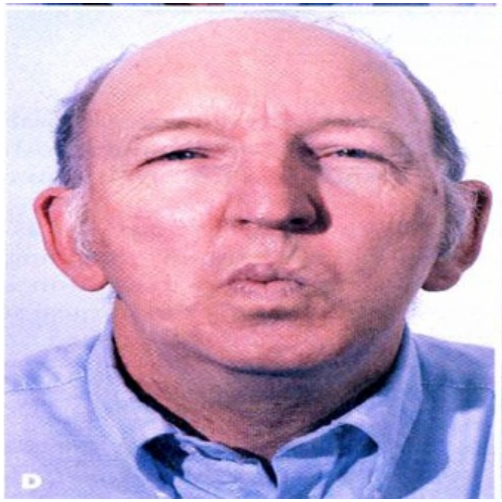


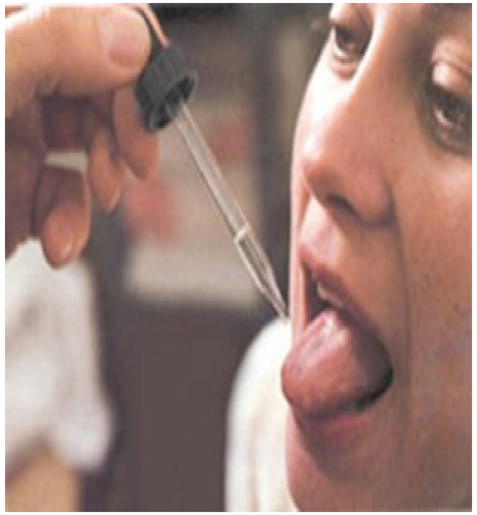


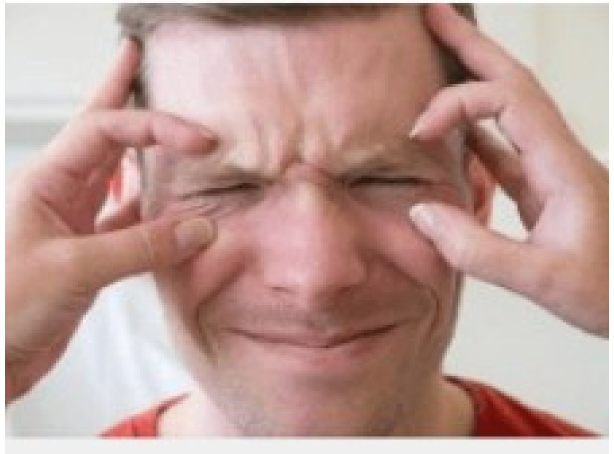
Facial nerve examination

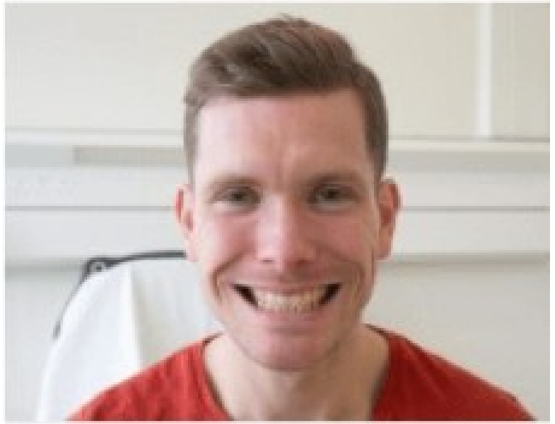




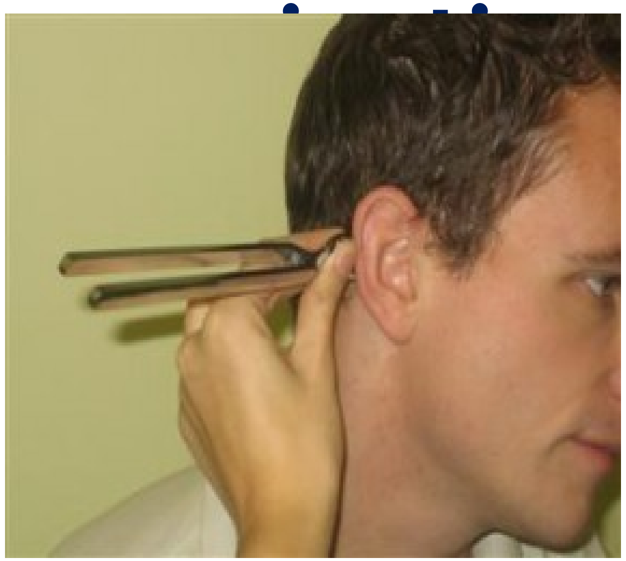




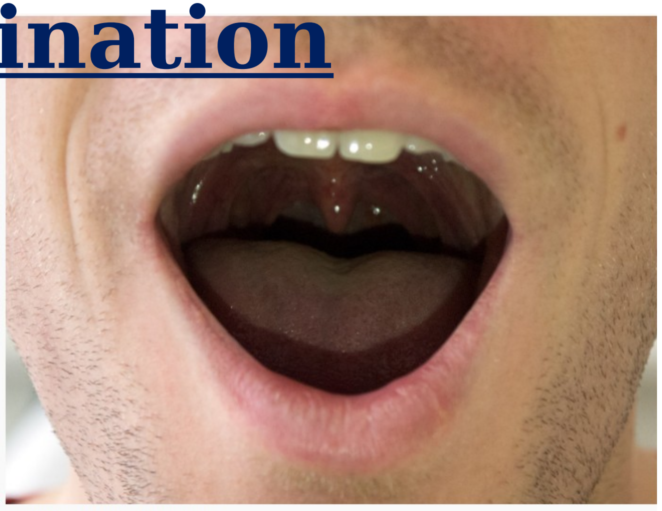


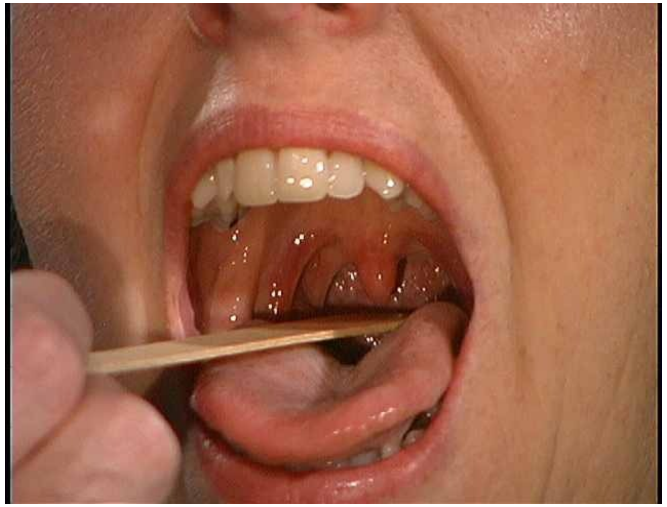


Vestibulo-cochlear nerve examination

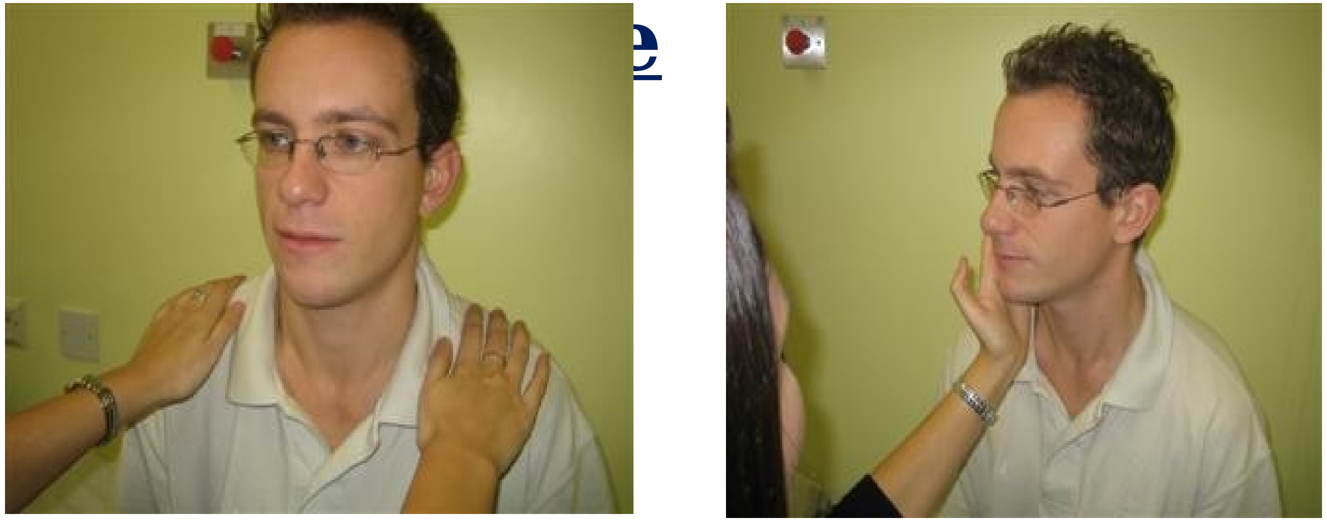


Glossopharyngeal and Vagal nerves examination

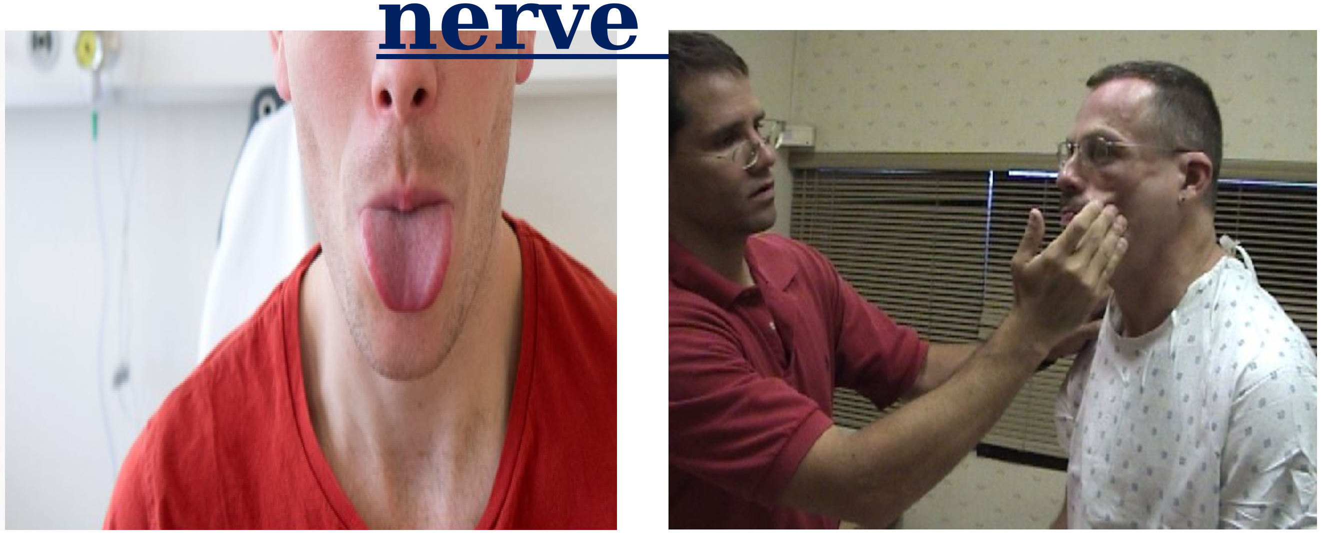




Accessory e



Hypoglossal nerve

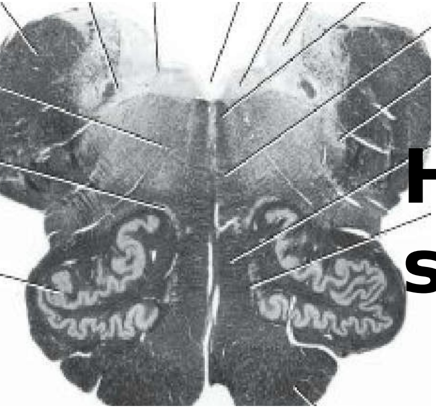


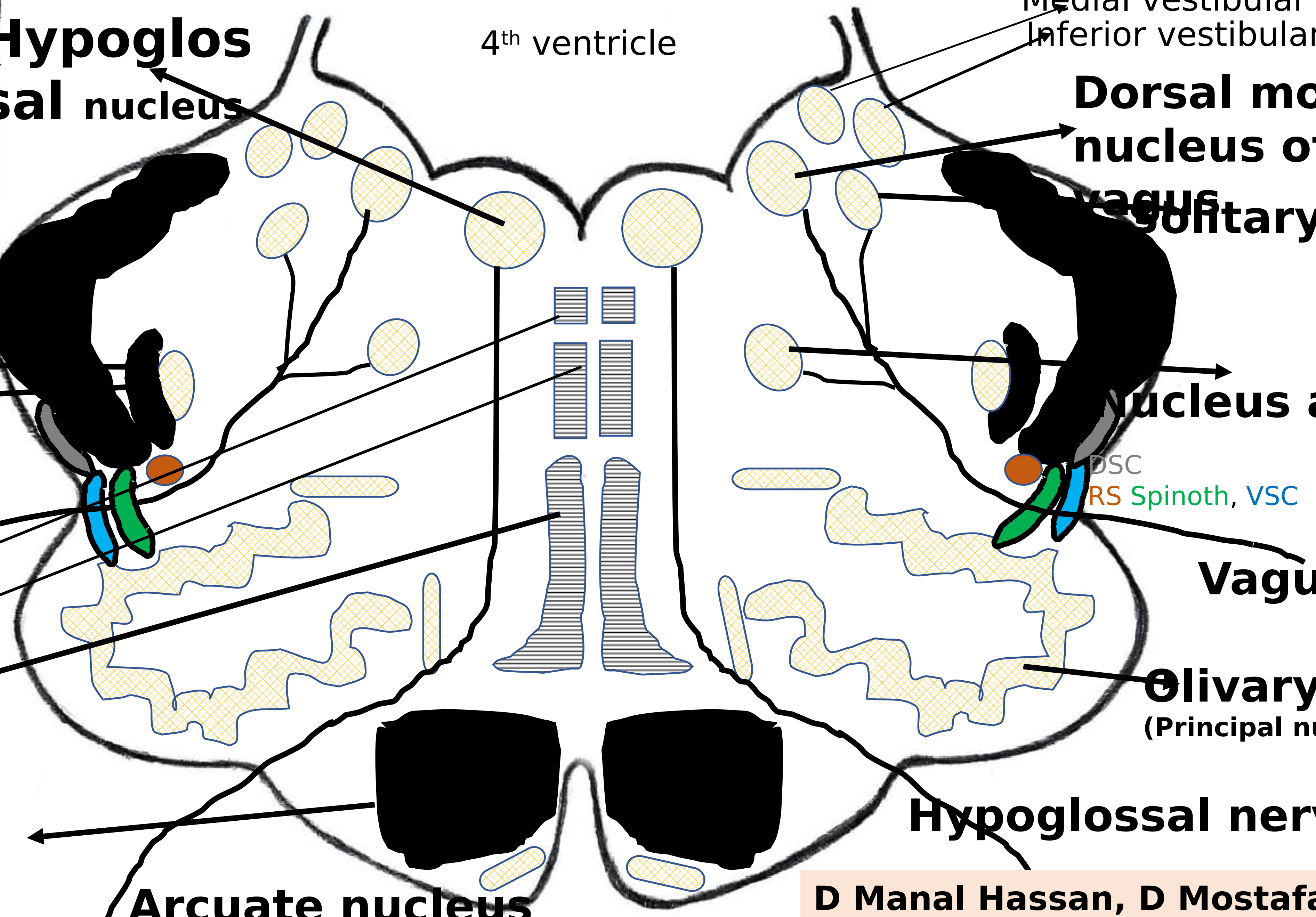
Mouira has her mouth deviated to the right and when she was asked to protrude her tongue, it was deviated to the left.
Deviation of the angle of mouth to the right side with ability to close her eyes

What are the cranial nerves affected in Monira and on which side?

5- Mention the site of the facial and hypoglossal nuclei in the brain stem

Open Medulla -- Weigert Pal





4th ventricle

Medial vestibular nucleus

Inferior vestibular nucleus

Hypoglos sal nucleus

Dorsal motor nucleus of vagus

Solitary nucleu

ICR

Spinal tract and nucleus of trigeminal V

Nucleus ambigu

DSC

RS Spinoth, VSC

MLB

Tectospinal

Vagus nerv

Medial lemniscus

Olivary nucleu

(Principal nucleus)

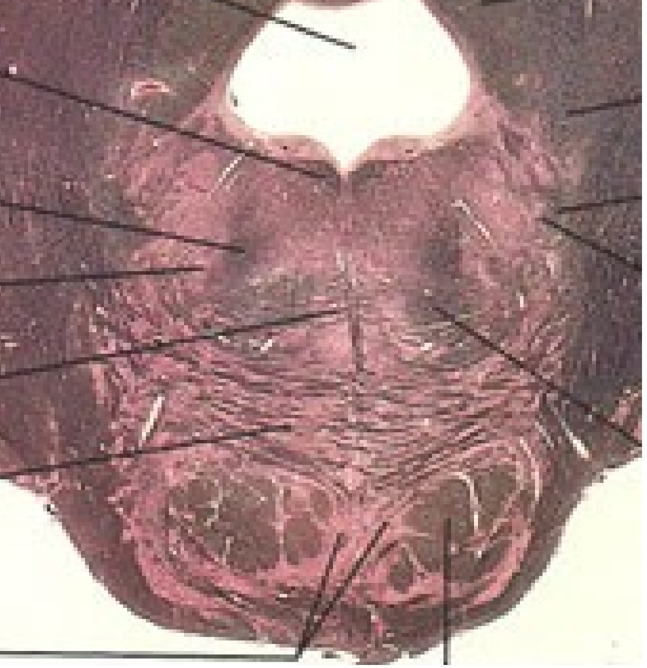
Pyramid

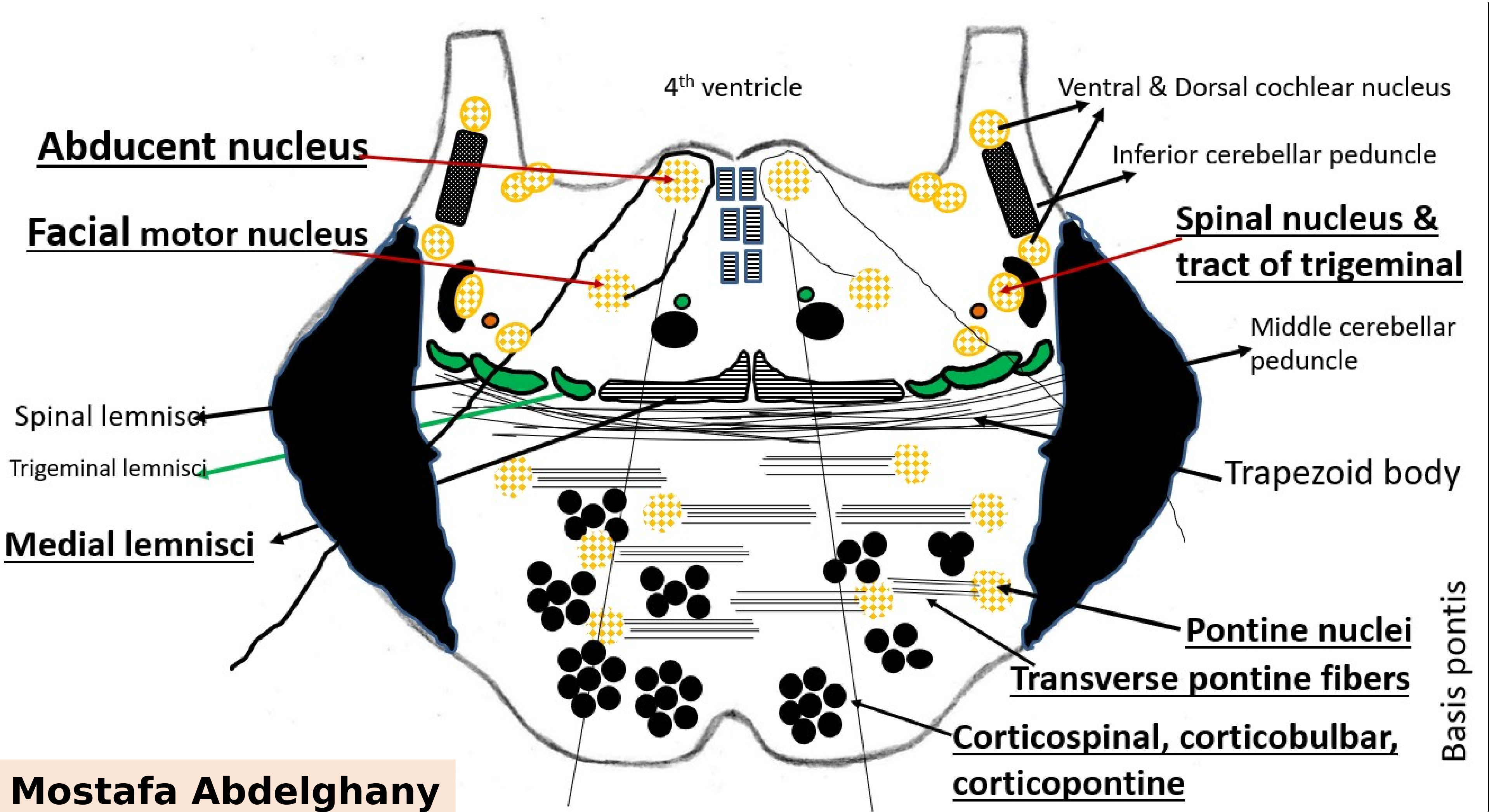
Hypoglossal nerve

Arcuate nucleus

D Manal Hassan, D Mostafa Abdelgh

Lower pons

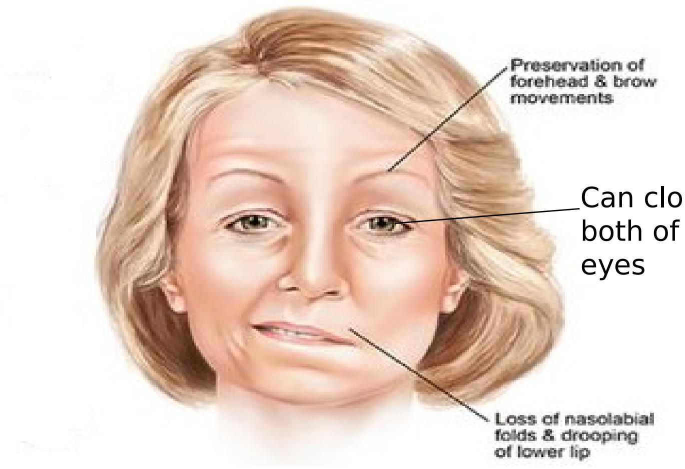



4th ventricle
Ventral & Dorsal cochlear nucleus
Inferior cerebellar peduncle
Abducent nucleus
Facial motor nucleus
Spinal nucleus & tract of trigeminal
Middle cerebellar peduncle
Spinal lemnisci
Trigeminal lemnisci
Trapezoid body
Medial lemnisci
Pontine nuclei
Transverse pontine fibers
Corticospinal, corticobulbar, corticopontine
Basis pontis

D Manal Hassan, D Mostafa Abdelghany

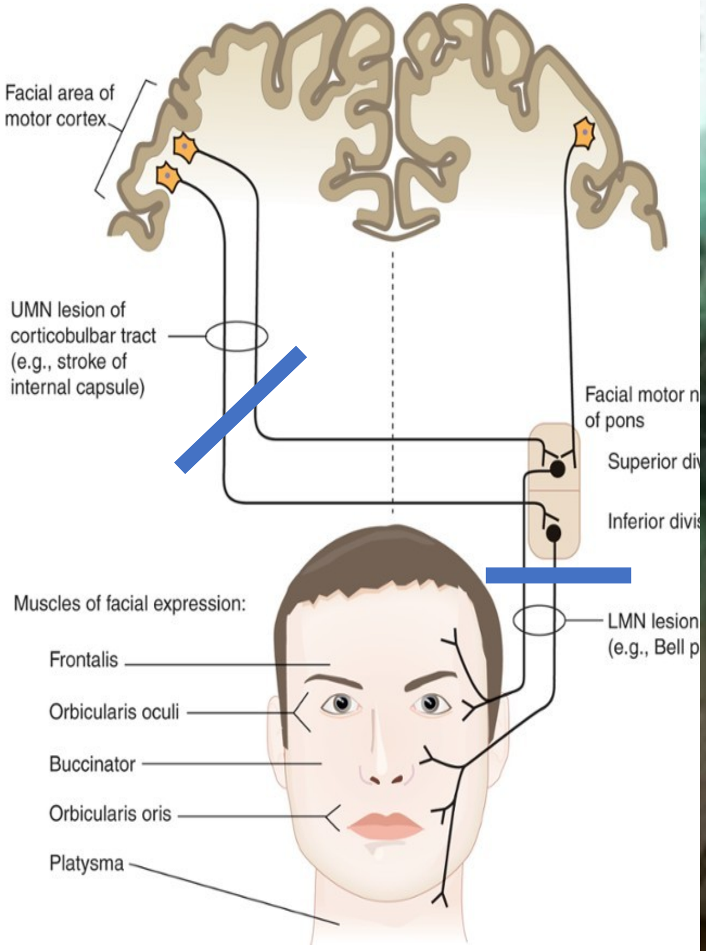
6- Does Monira have upper or lower motor neuron lesion of facial….. why?

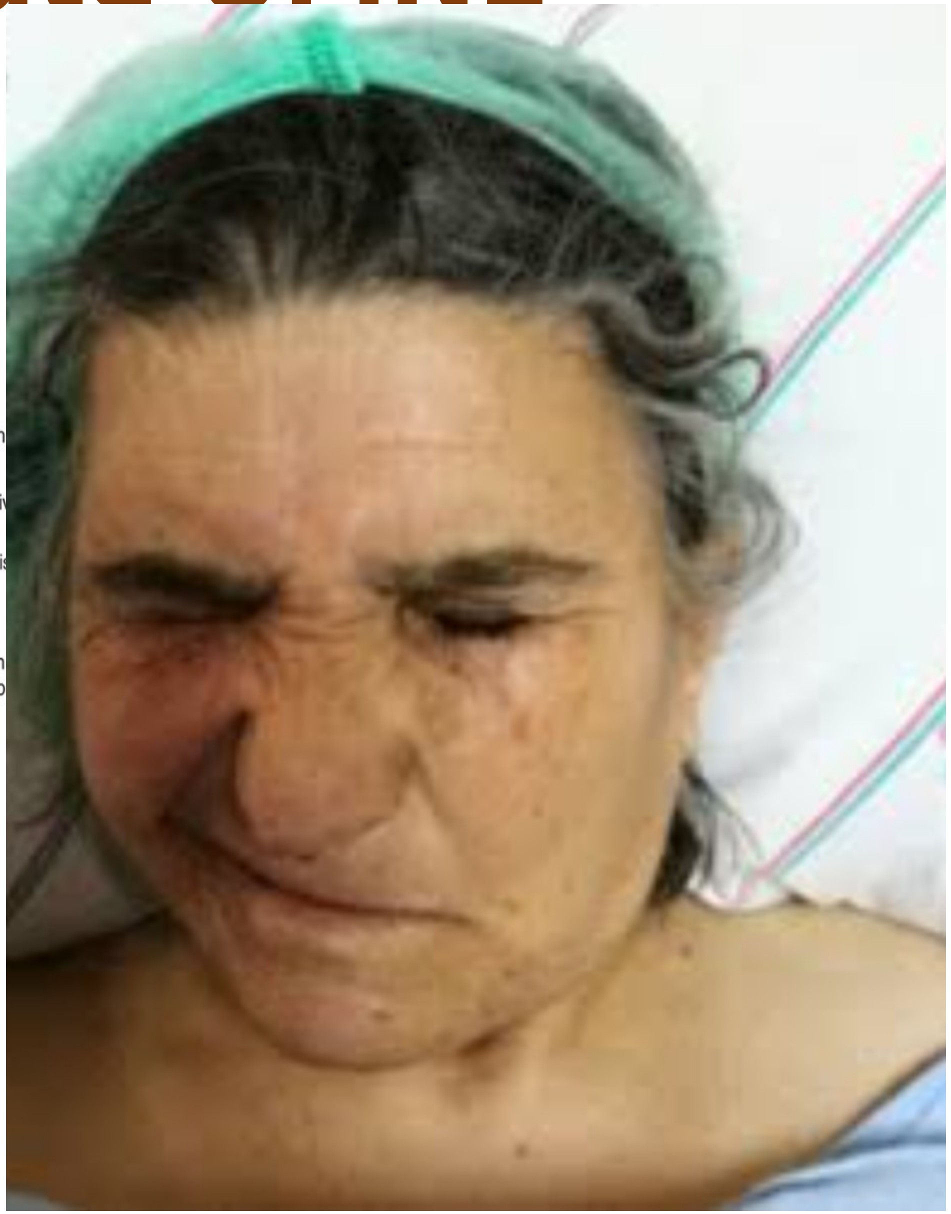
How could you differentiate UMNL from LMNL in


Preservation of forehead & brow movements
Loss of nasolabial folds & drooping of lower lip

Can close both of her eyes

In UMNL of facial nerve: lower quadrant of face on **opposite** side is affected.





Definition. A type of primitive reflex that is elicited by **repetitive tapping on the forehead**. Normal subjects usually blink in response to the first several taps, it is *lost in lower motor fascial pulsy*

7- A-What is the part of CNS affected in Monira ?

B- What are structures passing in this area?

Internal capsule

- **It consists of:**

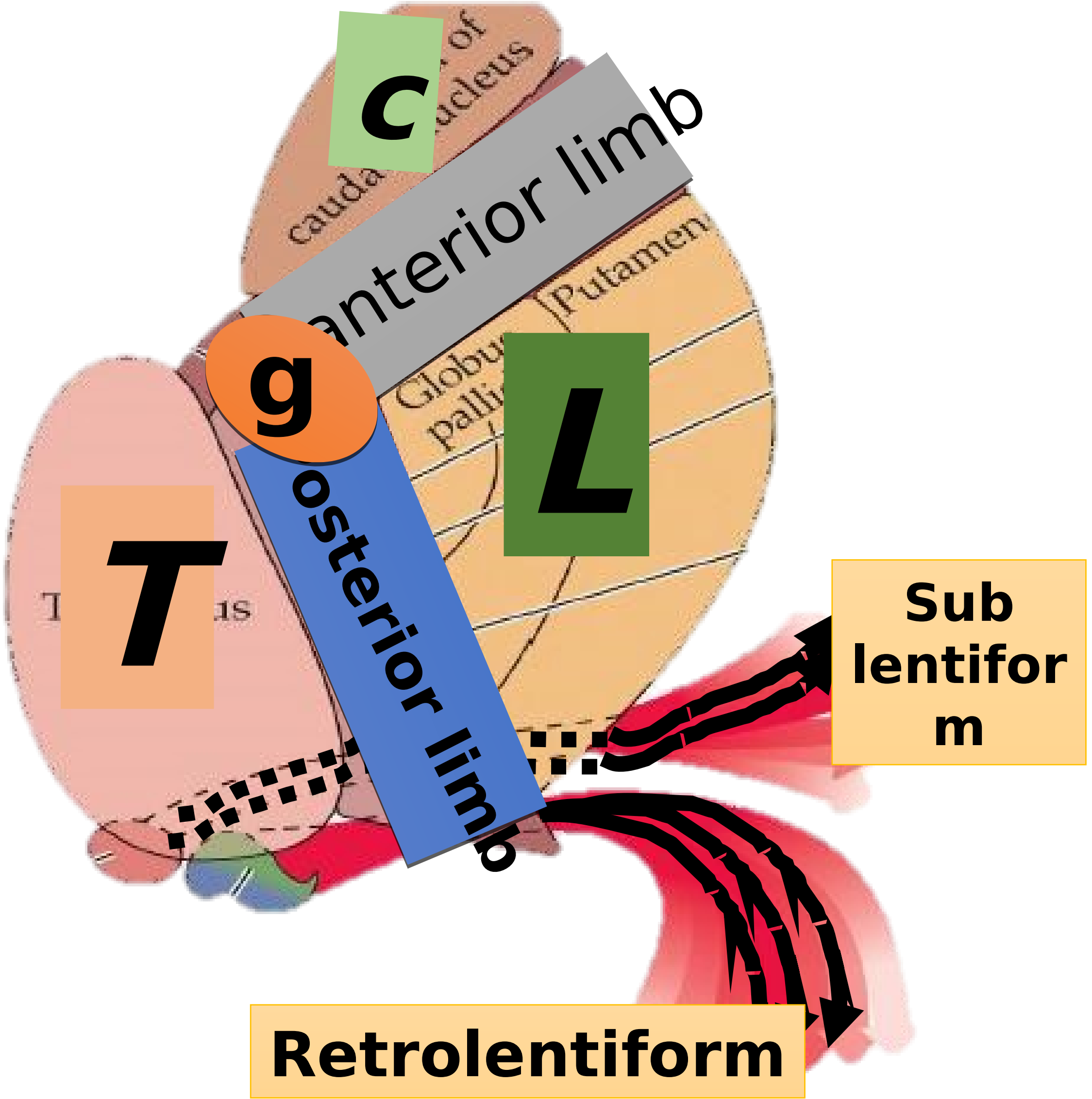
I. Anterior limb

II. Genu

III. Posterior limb

IV. Retrolentiform part

V. Sublentiform



Anterior limb

1. ***Frontopontine fibers:***
2. ***Anterior Thalamic***

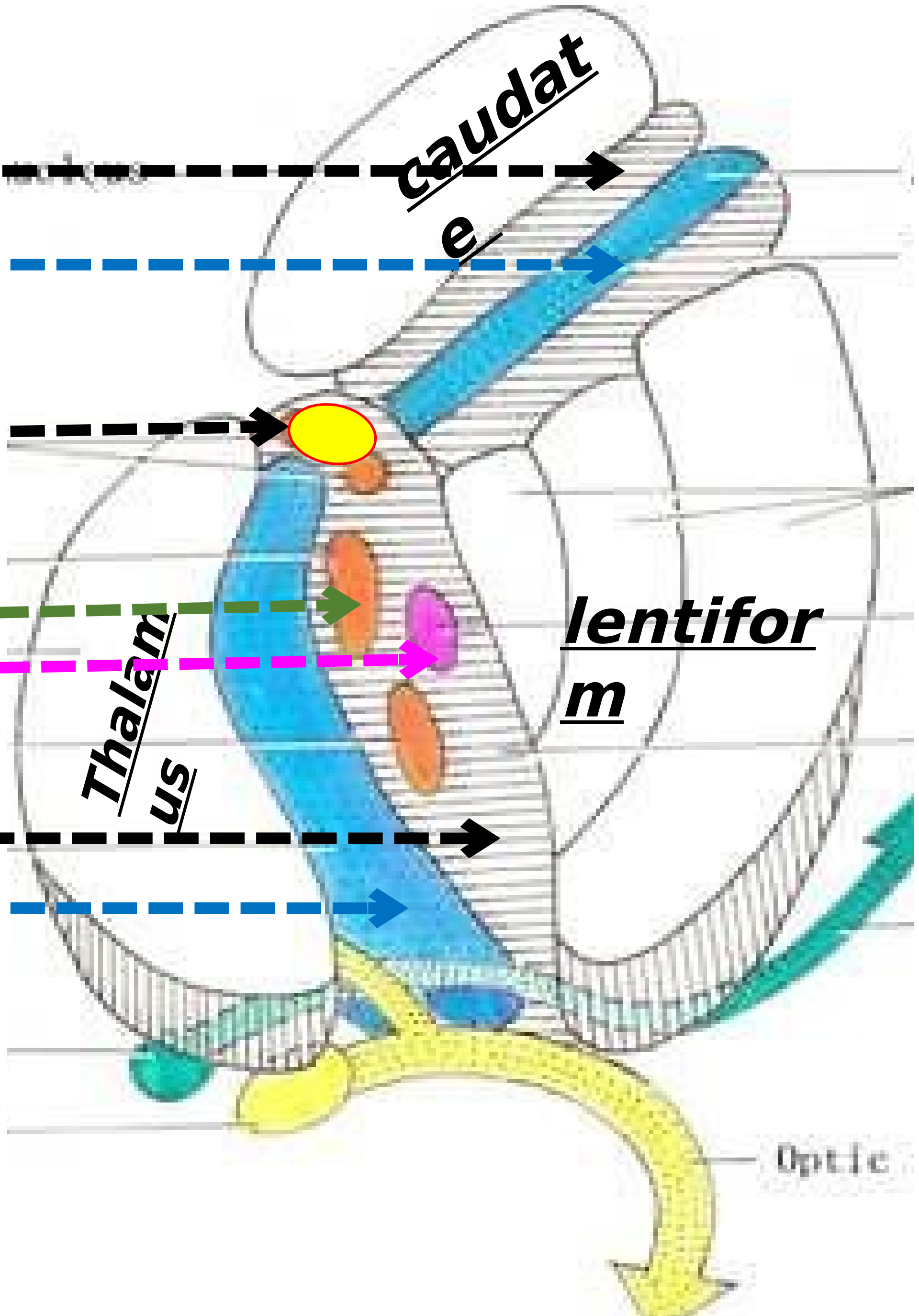
Genu

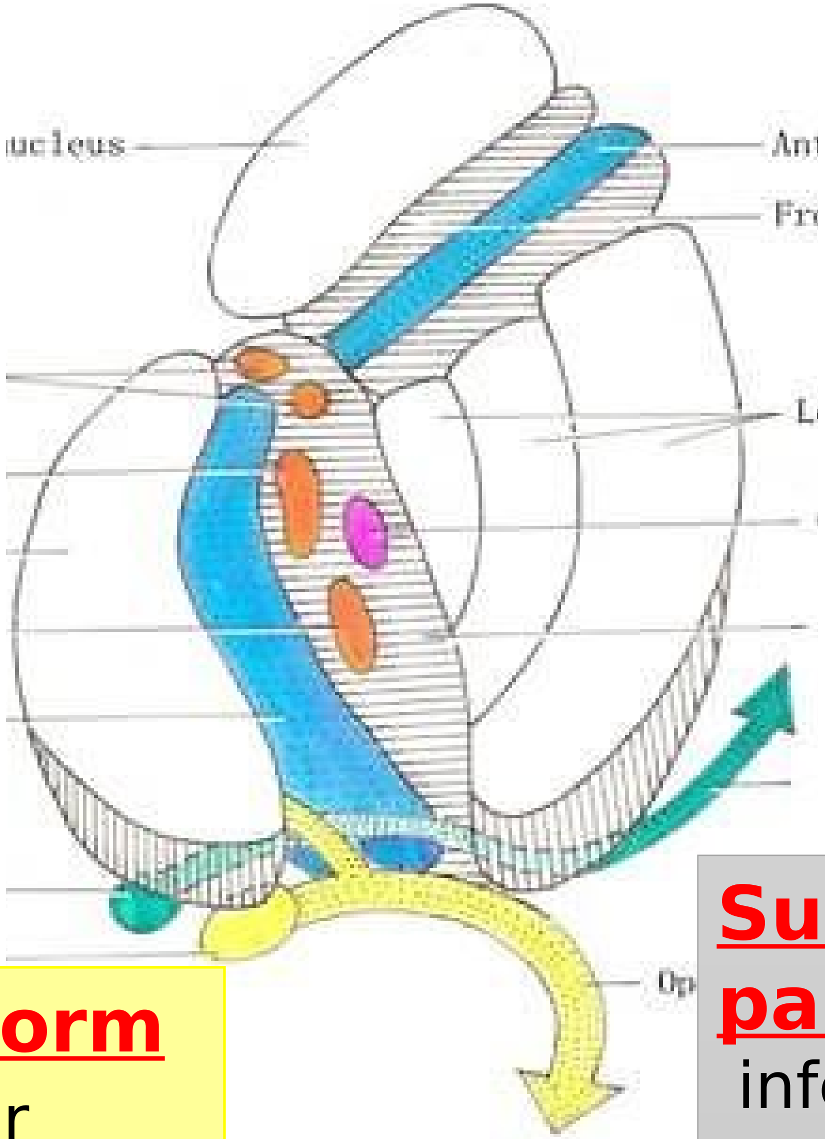
Contains ***corticonuclear fibers***

Posterior limb

1. ***Corticospinal fibers***
2. ***Corticorubral fibers***

3. ***Frontopontine fibers***
4. ***Superior thalamic radiation***





Retrolentiform part Posterior thalamic R.
= **(optic radiation)**
(occipito-pontine

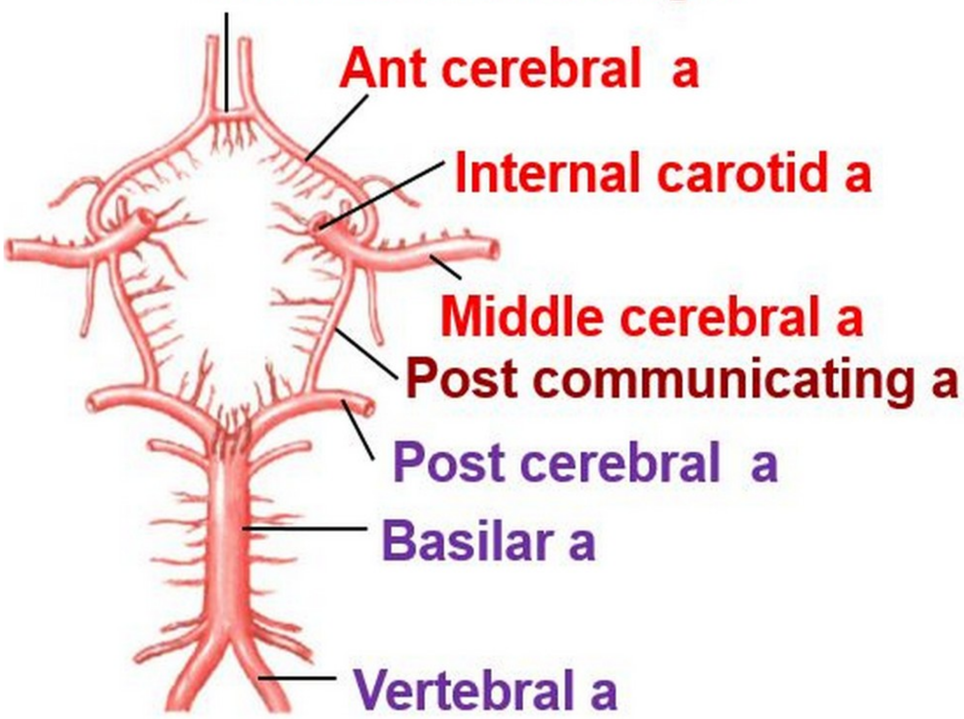
Sublentiform parts
inferior thalamic R.

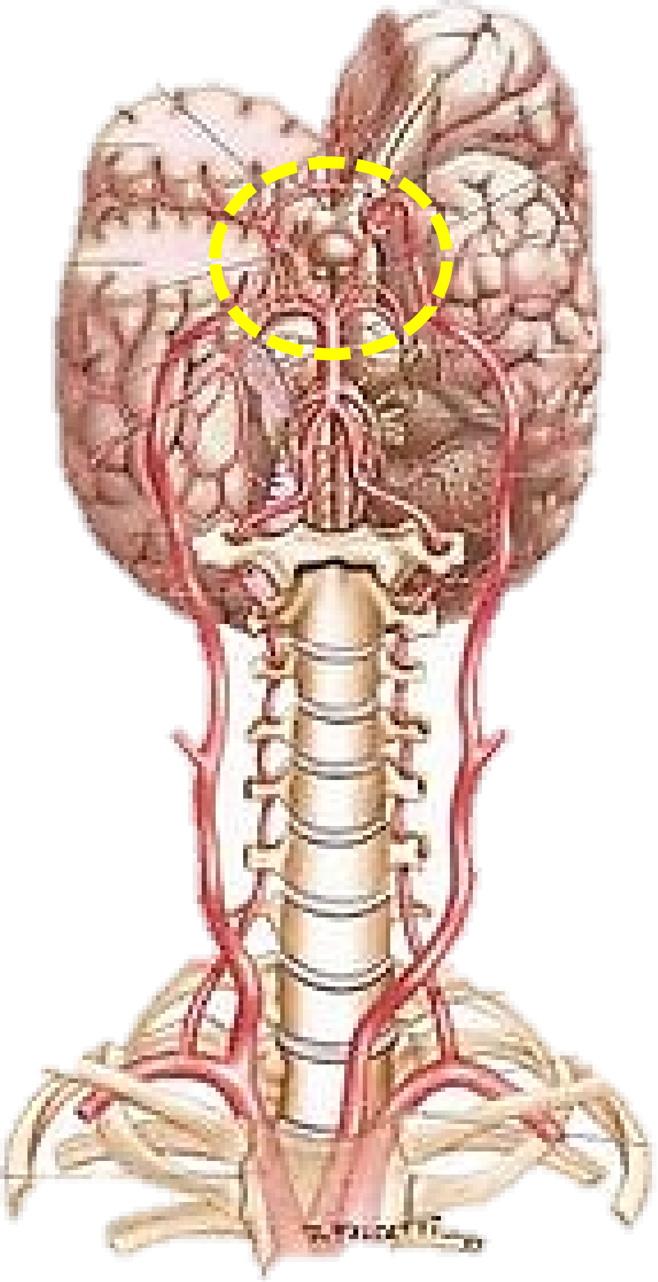
= **auditory radiation**
(Temporo-pontine

8- What are the blood vessels affected in Monira?

Blood supply of internal capsule

Central branches of
Anterior & Middle cerebral artery





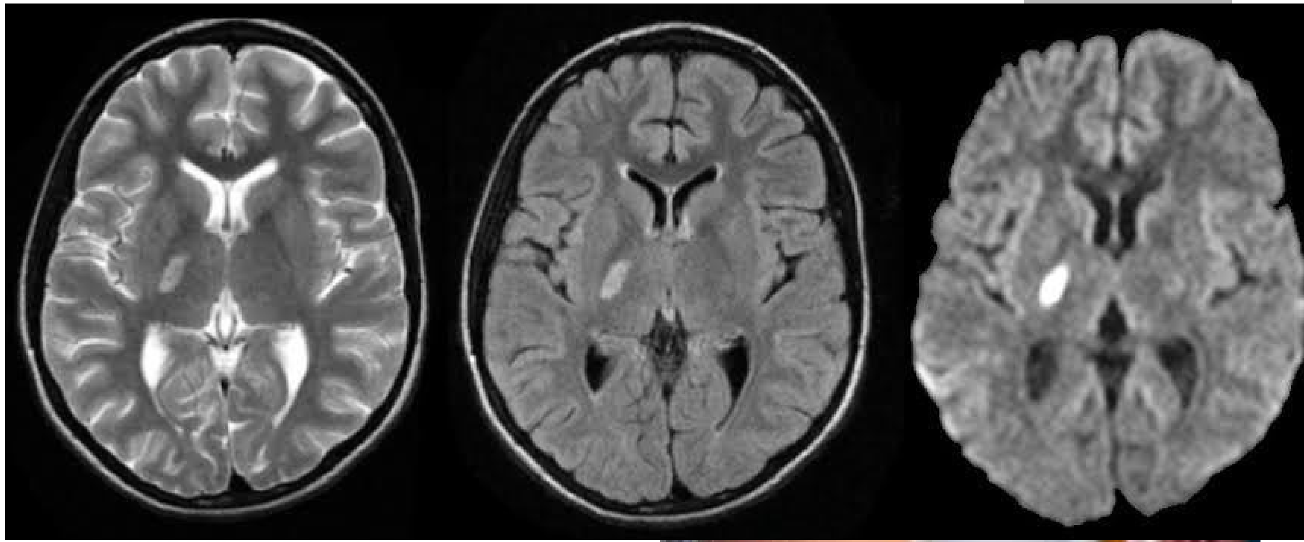
9- What is the first step that should be done to Monira?

Welcome Back

I am unable to walk

The neurologist urgently asked for

CT brain… Why?

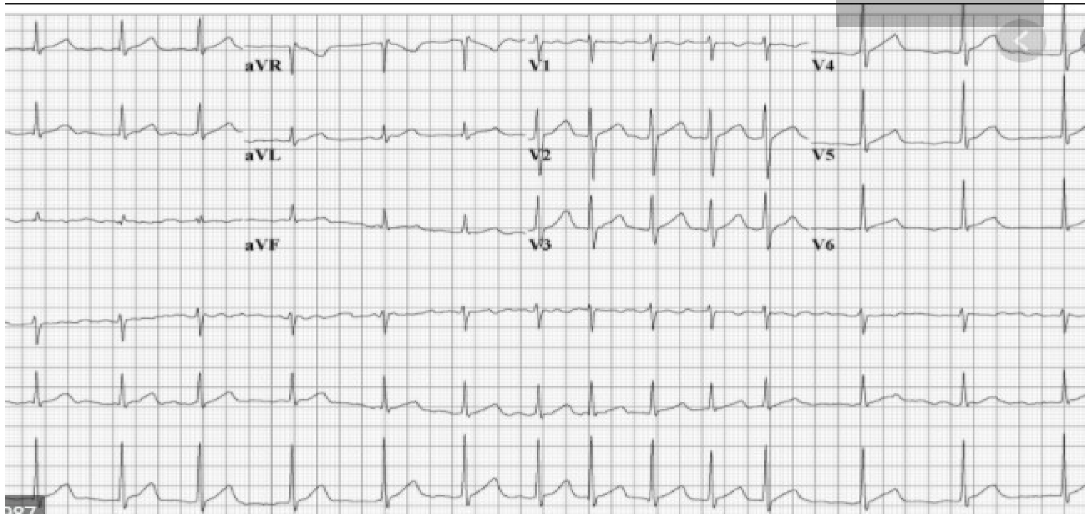


RTPA was started immediately.

A Blood sample was taken for examin

- **CBC**
- **PT/INR**
- **HbA1C**
- **Lipid profile**
- **Serum creatinine**

I am unable to walk



- Echo and carotid doppler done to assess risk factors for stroke.
- ECG shows *AF*

Two days later, the neurologist referred Monira to a physiotherapy specialist who told Monira that he should have **3 sessions of physiotherapy/week**.

I am unable to walk

On the day of hospital departure, the neurologist told Monira that he has to take

- **Aspocid 75mg daily**
- **Clopidogrel**
- **Oral anticoagulants**
- **Simvastatins**
- **Antihypertensives**
- **Antidiabetic**

- *Antiplatelet vs anticoagulants*

Discussion

The doctor gave Monira immediate rTPA

10- a. Why?

b. What is the mechanism of action of RTPA

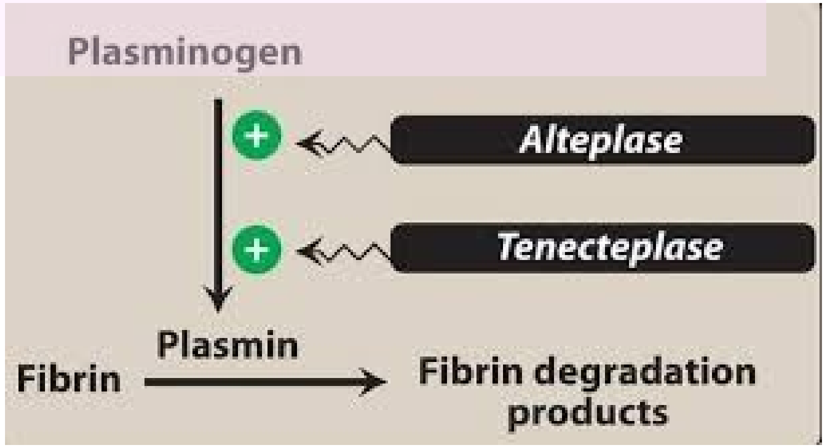
c. What are its advantages?

The doctor gave Monira immediate rTPA

Why?

- immediate steps to save the patient’s life by dissolving the blood clot and restore the blood flow
- In general, venous thrombi are lysed more easily by fibrinolytics than arterial, and recent thrombi respond better

What is the mechanism of action of RTPA



What are its advantages?

- Fibrin specific
- Less intracranial bleeding
- Dose not cause allergic reaction (non antigenic)

On the day of hospital departure, the neurologist told Medhat that he has to take

- Aspirin 75mg daily
- oral anticoagulants

11 –Explain why Mr Medhat has to take oral anticoagulants?

Stroke prevention in atrial fibrillation

- **Oral anticoagulation** is the therapy of choice for primary and secondary stroke prevention in patients with atrial fibrillation and any of the known additional risk factors.

- Asymptomatic patients younger than 65 years with atrial fibrillation and none of the other risk factors are at a low risk and either should be treated with aspirin or should not be treated at all.

Guidelines for secondary prevention(AHA/ASA)

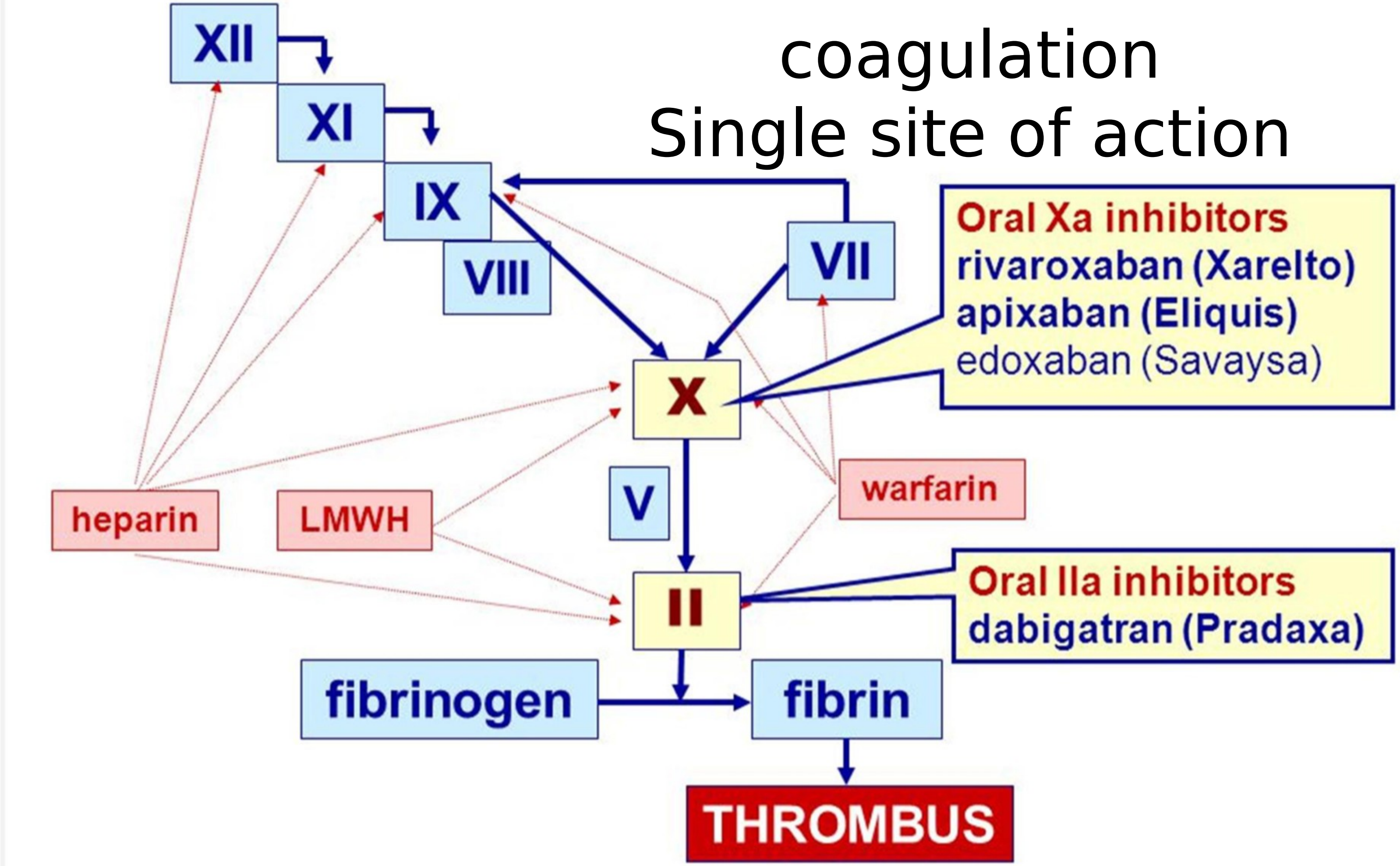
- Non-cardioembolic ischemic stroke
  - Antiplatelet agents rather than oral anticoagulation
    - aspirin monotherapy
    - combination of aspirin and extended-release dipyridamole,
    - clopidogrel monotherapy are all acceptable options for initial therapy
    - For patients who have an ischemic cerebrovascular event while taking aspirin
      - the combination of aspirin and extended-release dipyridamole is recommended over aspirin alone (*IB*)

On the day of hospital departure, the neurologist told Medhat that he has to take

- Aspirin 75mg daily
- Oral anticoagulants

- **Mechanism of action of available oral anticoagulant?**
- **What is the advantages of using new oral antigcoagulants over other anticoagulant**

New oral anticoagulant: Direct inhibition of coagulation
Single site of action



Oral Xa inhibitors
rivaroxaban (Xarelto)
apixaban (Eliquis)
edoxaban (Savaysa)

Oral IIa inhibitors
dabigatran (Pradaxa)

Advantages of NOAC over warfarin:
Immediate onset and rapid offset
The rates of intracranial hemorrhage are lower for NOACs compared with warfarin (safer therapy)
Fewer interaction so predictable response
No need for frequent monitoring.

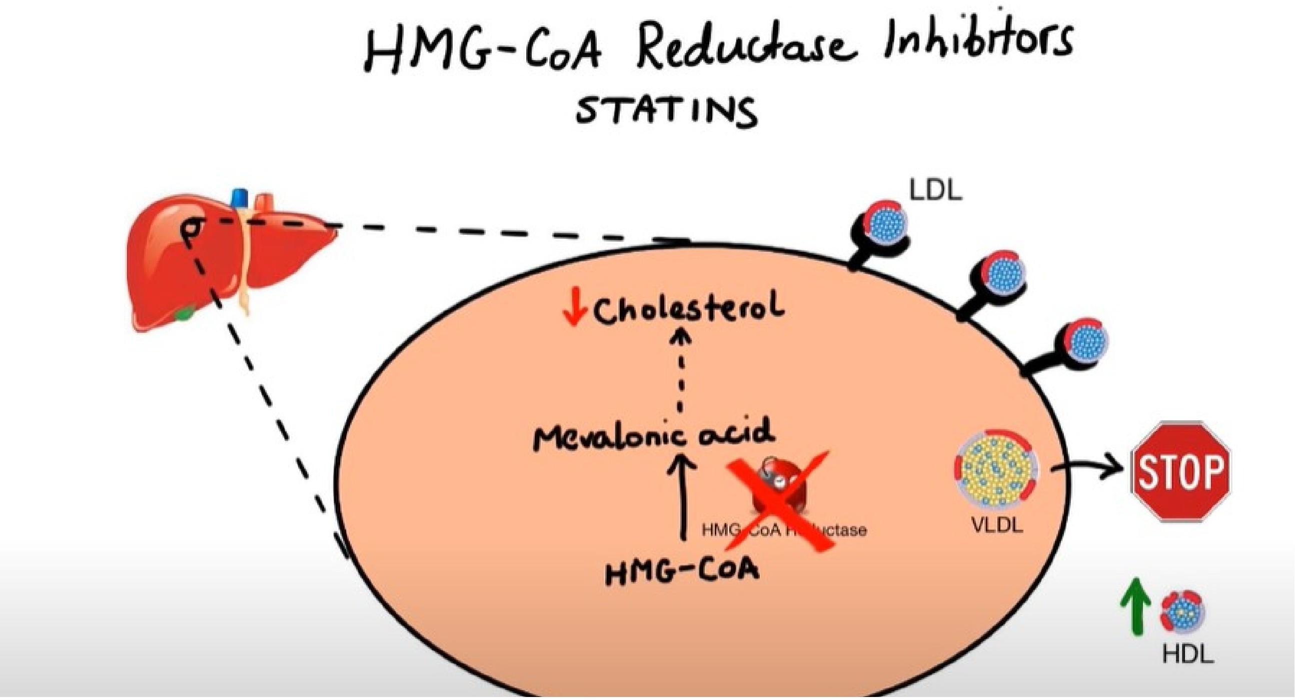
NB: Dabigatran carries a higher risk of gastrointestinal bleeding compared with warfarin, and it appears to increase the risk of myocardial infarction

On the day of hospital departure, the neurologist told Medhat that he has to take

- statin

12- What is the mechanism and side effects statin?

HMG-CoA Reductase Inhibitors (Statins) (Simvastatin)



+ stop VLDL release and ↑HDLCardiopreotective: vasodilators- □ atherosclerosis (stabilize plaque)

- Structural analogs of HMG-CoA
- inhibit HMG-CoA reductase (rate limiting)

□ Cholesterol synthesis in liver

- compensatory □ synthesis of LDL receptor

□ uptake of LDL in liver

□ plasma LDL-cholesterol.

Statin side effects

- **HMG-C**o **A** **R**eductase **I**nhibitors
  - **H**epatotoxic increase liver enzymes
  - **M**yopathy
  - **G**I effects
  - **C**ataract
  - **R**habdomyolysis
  - **I**ncrease risk of diabetes

Monira should have **3 sessions of physiotherapy/week**.

Why ?

Take home message

To perform neurological examination

- **Mental status**
- **Posture, Gait**
- **Motor examination**
- **Sensory examination**
- **Cranial nerve examination**

- **Don’t decrease the blood pressure in a case of acute thrombosis.**

Thank you